Evaluation and Management of the Physiologically Difficult Airway: Consensus Recommendations From Society for Airway Management

Rebecca L. Kornas, MD,* Clark G. Owyang, MD,† John C. Sakles, MD,‡ Lorraine J. Foley, MD, MBA,§ and Jarrod M. Mosier, MD,‡‖ on behalf of the Society for Airway Management's Special Projects Committee

Multiple international airway societies have created guidelines for the management of the difficult airway. In critically ill patients, there are physiologic derangements beyond inadequate airway protection or hypoxemia. These risk factors contribute to the "physiologically difficult airway" and are associated with complications including cardiac arrest and death. Importantly, they are largely absent from international guidelines. Thus, we created management recommendations for the physiologically difficult airway to provide practical guidance for intubation in the critically ill. Through multiple rounds of in-person and telephone conferences, a multidisciplinary working group of 12 airway specialists (Society for Airway Management's Special Projects Committee) over a time period of 3 years (2016–2019) reviewed airway physiology topics in a modified Delphi fashion. Consensus agreement with the following recommendations among working group members was generally high with 80% of statements showing agreement within a 10% range on a sliding scale from 0% to 100%. We limited the scope of this analysis to reflect the resources and systems of care available to out-of-operating room adult airway providers. These recommendations reflect the practical application of physiologic principles to airway management available during the analysis time period. (Anesth Analg 2021;132:395–405)

GLOSSARY

A-a = alveolar-arterial; **ARDS** = acute respiratory distress syndrome; **BVM** = bag-valve-mask; **COVID-19** = coronavirus disease 2019; **DSI** = delayed sequence intubation; **ECMO** = extracorporeal membrane oxygenation; **ED** = emergency department; **EtN_2** = end-tidal nitrogen; **EtO_2** = end-tidal oxygen; **FRC** = functional residual capacity; **HFNO** = high-flow nasal oxygenation; **ICP** = intracranial pressure; **ICU** = intensive care unit; **LPM** = liters per minute; **LV** = left ventricle; **NC** = nasal cannula; **MAP** = mean arterial pressure; **NIPPV** = noninvasive positive pressure ventilation; **NMBA** = neuromuscular blocking agent; **NRM** = nonrebreather mask; **OR** = operating room; **PEEP** = positive end-expiratory pressure; **PIH** = peri-intubation hypotension; **RAAS** = renin-angiotensin-aldosterone system; **RV** = right ventricle; **SAM** = Society for Airway Management; **SD** = standard deviation; **SARS-CoV-2** = severe acute respiratory syndrome coronavirus 2; **SI** = shock index; **SpO_2** = peripheral arterial oxygen saturation; **V/Q** = ventilation/perfusion

Many societies have published guidelines on airway crisis management or resource allocation; however, there are few recommendations on the physiologic considerations for airway management.[1–4] The Society for Airway Management (SAM) was founded in 1995 as an interdisciplinary forum for physicians and nonphysicians from 28 different countries who specialize in airway management. A goal of SAM is to contribute to the scientific advancement of airway management through representation from multiple specialties; these recommendations aim to fill this void by creating physiologically directed recommendations sourced from a multidisciplinary group of providers.

Annually, there are over 141 million emergency department (ED) visits in the United States with nearly 2 million requiring intensive care unit (ICU) admission.[5] Many of these patients require tracheal

From the *Department of Emergency Medicine, Denver Health, Denver, Colorado; †Division of Pulmonary and Critical Care Medicine, Department of Medicine, Weill Cornell Medicine, New York Presbyterian Hospital, New York, New York; ‡Department of Emergency Medicine, University of Arizona College of Medicine, Tucson, Arizona; §Department of Anesthesiology, Winchester Hospital, Tufts University School of Medicine, Boston, Massachusetts; and ‖Division of Pulmonary, Allergy, Critical Care and Sleep, Department of Medicine, University of Arizona College of Medicine, Tucson, Arizona.

Accepted for publication September 3, 2020.

DOI: 10.1213/ANE.0000000000005233

Funding: None.

Conflicts of Interest: See Disclosures at the end of the article.

Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website (www.anesthesia-analgesia.org).

R. L. Kornas and C. G. Owyang contributed equally and share first authorship.

A full list of contributors can be found at the end of the article.

Reprints will not be available from the authors.

Address correspondence to Clark G. Owyang, MD, Weill Cornell Medicine, 1305 York Avenue, Y-1047, Box 96, New York, NY, 10021. Address e-mail to clark.owyang@gmail.com.

intubation, which is the third most commonly performed procedure in US hospitals,[6] and carries high complication rates ranging from transient oxygen desaturation and hypotension to cardiovascular collapse and death.[7,8]

Physiologic derangements predominantly drive these risks with intubation in this population and often precipitate complications despite first attempt success. These pathophysiologic alterations limit the ability to preoxygenate, to maintain oxygenation during intubation, or to tolerate the transition to positive pressure ventilation. Collectively, these are commonly referred to as the "physiologically difficult airway."[9,10] Iatrogenic alterations to patient physiology through induction agents and resuscitation strategies contribute additional risk. The Special Projects Committee of the SAM developed the following list of recommendations to improve the safety of physiologically difficult airway management.

METHODS

Over a 3-year time period (September 2016 to September 2019), 12 members of the Society for Airway Management's Special Projects Committee representing Anesthesia, Critical Care Medicine, and Emergency Medicine developed recommendations for management of the physiologically difficult airway. Members of the Special Projects Committee performed study selection via a convenience sample after extensive literature review, and a representative working group searched relevant literature from various subspecialty backgrounds.

We used a modified Delphi method to develop a list of recommendations provided by each of the working group members. We then conducted an anonymous electronic survey via Qualtrics, where each of the members assigned their level of agreement with each recommendation on a visual analog scale ranging from 0% (complete disagreement) to 100% (complete agreement). The 12-member working group's recommendations and level of agreement are provided below and in Supplemental Digital Content, Tables, http://links.lww.com/AA/D206. Results are reported as mean and standard deviation. No formal risk of bias evaluation was performed.

RECOMMENDATIONS

Hypoxemia

Preoxygenation. In hypoxemic respiratory failure, there is a failure to maintain adequate arterial oxygenation, most commonly due to shunt and ventilation/perfusion (V/Q) mismatch. Critically ill patients are at high risk for rapid desaturation because of reduced functional residual capacity (FRC), V/Q mismatch, and shunt—all of which can be acutely worsened on induction.[9] Subsequently, there is an increased risk for arrhythmia, hemodynamic instability, anoxic brain injury, and even cardiopulmonary arrest.[11,12] While there was complete agreement among the Special Projects Committee that patients with hypoxemic respiratory failure are at high risk of desaturation during intubation (100% agreement [standard deviation {SD} 0%]), there was not complete agreement that desaturation is the biggest risk factor for cardiopulmonary arrest (87% agreement [SD 29%]) (Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206). All members agreed that reduced FRC, atelectasis, alveolar filling, shunt physiology, and increased dead space all contribute to difficulty with adequate preoxygenation (100% agreement [SD 0%]; Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206).

Optimizing preoxygenation to extend the time to desaturation improves the likelihood of first-pass success.[13] Because desaturation occurs in 19%–70% of intubations and is the most common reason to abort first attempt at intubation, prioritization of preoxygenation, and apneic oxygenation are prudent in all patients.[7,14–17] Historically, preoxygenation was achieved using a nonrebreather mask (NRM) without a leak delivering 100% oxygen over 3–5 minutes of tidal volume breathing or 8 vital capacity breaths (Table).[18] The end points of maximal preoxygenation and denitrogenation are end-tidal oxygen (EtO_2) concentration of approximately 90% and end-tidal nitrogen (EtN_2) concentration of 5%.[19,20] When a NRM lacking a tight seal is used, there is a leak around the edges of the mask and patients mix the supplied oxygen with entrained ambient air, decreasing the effective fraction of inspired oxygen. This is particularly relevant to critically ill patients with high minute ventilation who are likely to entrain large volumes of room air.

Recent studies have evaluated the efficacy of different methods of preoxygenation. Groombridge et al[21] found NRM mask at 15 liters per minute (LPM) less effective than bag-valve-mask (BVM) or a closed anesthetic circuit. Driver et al[22,23] showed that increasing the oxygen flow by using flush rate (oxygen flowmeter turned maximally up ~ 50–70 LPM) through a standard NRM was not inferior to BVM preoxygenation at 15 LPM or flush rate. Of note, if a mask leak is present, the addition of a nasal cannula (NC) at 10 LPM is helpful in improving EtO_2.[24] Patients should be preoxygenated using a NRM at flush rate as well as a NC at 15 LPM allowing for continued apneic oxygenation,[21,24] and physiologic end points (eg, EtO_2) should be implemented to improve practice.

Preoxygenation is closely linked with the concept of apneic oxygenation, which serves to continuously replenish the oxygen consumed from the FRC during

Table. Top 10 Society for Airway Management Key Recommendations for Evaluation and Management of the Physiologically Difficult Airway

Physiology	Recommendations
Hypoxemia	1. Preoxygenation should be performed using high-flow oxygen for at least 3 min, or 8 vital capacity breaths. 99% agreement (SD 4.5%)
	2. Desaturation is the biggest risk factor for cardiopulmonary arrest. 87% agreement (SD 29%)
	3. If the patient has significant shunt physiology or reduced functional residual capacity (eg, pregnancy, obesity, ARDS), preoxygenation should be performed with PEEP using NIPPV. 98% agreement (SD 7.5%)
	4. Patients should be preoxygenated in the upright position when possible. 98% agreement (SD 6.3%)
	5. Delayed sequence intubation is an option for patients who cannot tolerate preoxygenation with NIPPV or HFNO. 100% agreement (SD 0%)
Hypotension	6. Risk factors for decompensation include vascular and cardiac effects of induction agents and effects of positive pressure ventilation. 99% agreement (SD 3%)
	7. Peri-intubation hypotension is independently associated with poor outcomes, including mortality, length of stay, and end-organ injury. 96% agreement (SD 12%)
	8. Patients should be screened for high risk of hemodynamic collapse with intubation. Those with a shock index >0.7 are at increased risk. 99% agreement (SD 1.5%)
	9. Fluid-responsive and fluid-tolerant patients should be fluid resuscitated before intubation, or at least during the intubation attempt. 99.5% agreement (SD 1.5%)
	10. When possible, vasopressor infusions should be started before intubation in patients that are not volume responsive or fluid tolerant. 99.5% agreement (SD 1.5%)

Abbreviations: ARDS, acute respiratory distress syndrome; HFNO, high-flow nasal oxygenation; NIPPV, noninvasive positive pressure ventilation; PEEP, positive end-expiratory pressure; SD, standard deviation.

apnea.[25,26] Apneic oxygenation has proven effective in prolonging safe apnea time in operating room (OR), prehospital, and ED studies.[27–31] Standard or wide-bore nasal prongs at 10–15 LPM are well tolerated, low-cost, and low-risk apneic oxygenation methods.[32] Use of capnography cannulas is discouraged because they will not achieve adequate apneic oxygenation given that they only allow for administration of effective oxygen flow rates of 0–6 LPM, often via cloud delivery while the nasal prongs sample exhaled gas.[32] High-flow nasal oxygen (HFNO) systems deliver humidified oxygen via proprietary NCs at 40–70 LPM, which increases apnea time and reduces the rate of carbon dioxide increase by gaseous mixing and flushing of dead space.[33]

While the individual studies vary, the benefits of apneic oxygenation will not rescue inadequate preoxygenation.[34] An ICU-based randomized controlled trial comparing 15 LPM apneic oxygenation via low-flow nasal oxygenation to usual care found no difference in median lowest arterial saturation or incidence of desaturation to <90% (45% vs 47%).[35] Notably, one-third of the patients in apneic oxygenation group were preoxygenated with NRM at 15 LPM, which is a poor method of preoxygenation.[21,22,24] Another randomized controlled trial of ED patients found no difference in the mean lowest saturation in the control (93%) and apneic oxygenation (92%) groups. These patients all had short periods of apnea, 64 seconds in the control group versus 58 seconds in the apneic oxygenation group. Importantly, these cases were rapid intubations with 90% patients successfully intubated in <100 seconds.[36] In this group of patients, it is likely that preoxygenation alone provided adequate oxygen reservoir to prevent desaturation (Supplemental Digital Content, Table 2, http://links.lww.com/AA/D206).[17]

Shunt physiology reduces the efficacy of preoxygenation with or without apneic oxygenation. In patients with shunt physiology (ie, a high alveolar-arterial [A-a] gradient), the FRC is less available to resaturate hemoglobin.[9,28,37] In this case, the partial pressure of arterial oxygen reflects the availability of the alveolar-capillary interface. Hemoglobin is typically saturated early in the transit across the alveolar-capillary interface, and excess oxygen is then dissolved in the blood down a concentration gradient. In a patient with acute respiratory distress syndrome, for example, a small volume FRC that is fully denitrogenated (EtO_2 >90%) and a Pao_2 of 100 reflects a patient at risk of rapid desaturation even if the oxygen saturation is >93% or the addition of apneic oxygenation because the oxygen reserve is not available to the pulmonary circulation. Reducing shunt physiology and improving V/Q mismatch becomes the priority, which is best accomplished with alveolar recruitment using a positive pressure strategy.

Noninvasive positive pressure ventilation (NIPPV) delivers a high oxygen concentration, unloads respiratory muscles, and recruits atelectatic alveoli.[37] Extraglottic devices are also useful for preoxygenation, especially if the patient requires higher airway pressures or cannot tolerate NIPPV mask.[38] Some of these devices have particularly high oropharyngeal seal pressures such as second-generation supraglottic airway devices (iGel >20 cm H_2O) and should be considered for preoxygenation in patients that have high positive end-expiratory pressure (PEEP) requirements (Supplemental Digital Content, Table 2, http://links.lww.com/AA/D206).[39] Derecruitment is possible when continuous positive pressure is removed, which should be considered in the intubation strategy.

HFNO may be an effective alternative to NIPPV for patients with shunt physiology who cannot achieve peripheral arterial oxygen saturation (SpO_2) > 93% on NRM at flush rate; who cannot tolerate a mask; or in whom an awake intubation is planned. These systems deliver continuous high-flow oxygen which increases end-expiratory volume and improves inspiratory flow dynamics, thus decreasing respiratory rate, reducing work of breathing, and improving thoracoabdominal synchrony. The constant concentration of oxygen delivered via HFNO is heated and humidified more closely approximating physiologic conditions than NIPPV. HFNO produces varying amounts of continuous positive pharyngeal pressure depending on flow rate, which provides a PEEP-like effect.[40,41]

Patients who do not achieve SpO_2 > 93% with a NRM at flush rate likely have significant shunt physiology. These patients should be transitioned to a positive pressure preoxygenation strategy using either NIPPV or assisted breathing through a BVM and a PEEP valve at 5–10 cm H_2O (Table),[42,43] or potentially HFNO (Table). When hypoxemia is refractory to high-flow oxygen and PEEP, inhaled pulmonary vasodilators may be useful adjuncts to improve V/Q mismatch and improve oxygenation. Inhaled pulmonary vasodilators improve hypoxemia by inducing local vasodilation of alveolar capillaries adjacent to well-ventilated areas of the lung and thereby improving V/Q mismatch. Finally, an awake intubation while maintaining spontaneous respiration may provide the safest approach for patients with refractory hypoxemia. These patients recruit dependent portions of the lung and have exaggerated pendelluft flow mediated through stress risers that is immediately lost on cessation of spontaneous breathing leading to worsening V/Q mismatch and often precipitous desaturation.[9]

Coronavirus disease 2019 (COVID-19), which has ravaged the globe in 2020, has presented a unique problem related to airway management. Patients present with often profound hypoxemia, most likely through alterations in V/Q mismatch induced by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) action on the renin-angiotensin-aldosterone system (RAAS) pathway. Given the high rate of health care worker transmission during the SARS epidemic, aerosol-generating procedures when necessary are to be performed thoughtfully in the context of emerging evidence.[44–46] Recommendations regarding aerosol-generating procedures and therapies limit NIPPV use (which vents directly into the room), acknowledge limited data on aerosols produced with HFNO, and discourage awake intubations.[47,48] Patients are generally preoxygenated with 15 LPM by NRM and intubated using rapid sequence induction, without apneic oxygenation, despite profound hypoxemia. As data begin to emerge on the risks of COVID-19 transmission with the use of NIPPV, HFNO, apneic, and awake intubation, more research is needed to optimize safe airway management while avoiding health care worker transmission.[46,49]

Critically ill patients should be preoxygenated in the 20°–30° upright position, when feasible (Table). OR studies show that preoxygenating patients in a head-up position increases the FRC, which improves preoxygenation and prolongs the safe apnea time.[50] A recent ICU-based randomized controlled trial by Semler et al[51] of ICU patients showed no improvement in oxygenation between groups preoxygenated in the head-up position versus supine position. However, most patients in this study had shunt physiology and the method of preoxygenation was not standardized, which may have limited the benefits of upright preoxygenation.

In combative or delirious patients, who are resistant to standard preoxygenation, ketamine, or a similar dissociative or nonrespiratory depressant sedative (eg, dexmedetomidine) can be considered to facilitate preoxygenation (Table). Once the patient is pharmacologically compliant, optimal preoxygenation can be performed before administration of the neuromuscular blocking agent (NMBA). In 1 small observational study, most patients had improved postsedation oxygen saturations[52]; however, intubation-related and patient-centered outcomes data are lacking. When performing this technique, a NMBA should be prepared and intubation equipment ready for immediate intubation before ketamine administration (Supplemental Digital Content, Table 2, http://links.lww.com/AA/D206).[53] Ketamine, while it generally preserves airway reflexes and spontaneous respirations, can induce apnea, laryngospasm, increased airway secretions, hypotension, and cardiac arrest.[54]

Recommendations

1. All patients should be maximally preoxygenated before intubation. 96% agreement (SD 12%)
2. Preoxygenation should be performed using high-flow oxygen for at least 3 minutes, or 8 vital capacity breaths. 99% agreement (SD 4.5%)
3. Maintenance of oxygenation during apneic period should be performed to prolong the duration of safe apnea. 100% agreement (SD 0%)
4. Apneic oxygenation can be performed with a standard NC at 15 LPM or HFNO systems at 40–70 LPM. 99% agreement (SD 3%)
5. If a tight-fitting NRM or NIPPV facemask is not available for preoxygenation, assisted spontaneous respirations with a BVM with a PEEP valve and 1-way exhalation valve should be used. 100% agreement (SD 0%)
6. If the patient cannot tolerate a tight-fitting NRM or NIPPV mask, heated HFNO systems with

40–70 LPM of flow should be used. 99% agreement (SD 4.5%)
7. If the patient has significant shunt physiology or reduced FRC (eg, pregnancy, obesity, acute respiratory distress syndrome [ARDS]), preoxygenation should be performed with PEEP using NIPPV versus BVM with a PEEP valve. 98% agreement (SD 7.5%)
8. Inhaled pulmonary vasodilators can be considered to improve ventilation-perfusion mismatch before intubation in patients with severe hypoxemia. 90% agreement (SD 18%)
9. When higher levels of PEEP are required, an extraglottic device should be considered for preoxygenation. 76% agreement (SD 40%)
10. Awake intubation to maintain spontaneous respiration should be strongly considered for patients with refractory hypoxemia. 95% agreement (SD 9%)
11. Patients should be preoxygenated in the upright position when possible. 98% agreement (SD 6.3%)
12. Ramped positioning should be performed when possible to improve grade of view, improve maintenance of oxygenation, and reduce aspiration risk. 99% agreement (SD 3.1%)
13. When delayed sequence intubation (DSI) is used, the operator should be ready for emergent intubation because the dissociative ketamine dose can be unreliable. 99.5% agreement (SD 1.5%)
14. Regarding DSI, we recommend using small doses of ketamine (10–20 mg aliquots) or dexmedetomidine to avoid apnea from a dissociative dose. 97% agreement (SD 7.5%)

Hypotension

Peri-intubation shock is independently associated with severe complications and death after emergency airway management.[55,56] As few as 10 minutes of hypotension can lead to poor outcomes in high-risk patients.[57] While awareness of peri-intubation hemodynamics has increased, there is no consensus definition of peri-intubation hypotension (PIH). Because there is no standard definition, the incidence of PIH varies widely from 0% to 44%.[58] Most definitions include any of the following in the 60-minute postintubation period: systolic blood pressure ≤90 mm Hg; mean arterial pressure ≤65 mm Hg; reduction in median systolic blood pressure of ≤20%; or any vasopressor administration.[59,60]

Risk factors for PIH include low mean arterial pressure (MAP) 60 minutes before intubation, preintubation shock index (SI, heart rate/systolic blood pressure) elevation, intubation for acute respiratory failure, advanced age, and chronic renal failure cormorbidities.[58,59] Associated with PIH, elevated SI (>0.8, normal SI 0.5–0.7) is associated with deteriorating cardiac performance, and can be an early sign of shock, indicating limited cardiovascular reserve during emergency airway management (Table).[61] Adverse events like organ dysfunction or death are associated with PIH in a dose-response manner, and quality improvement initiatives like intubation bundle checklists may improve the associated hypotension and hypoxemia.[16]

From the NEAR II data registry on 8937 ED intubations, cardiac arrest complications comprised 0.4% of all encounters.[62] A number of recent analyses have reinforced the importance of physiologic reserve and hemodynamic resuscitation in the peri-intubation time period. Multiple retrospective studies have shown the importance of preintubation SI and hypotension for significant association with peri-intubation hemodynamic collapse.[8,63–65] Similar to fluid responsiveness in the context of right ventricular (RV) failure, assessing volume status, increasing mean systemic filling pressure, and determining fluid responsiveness in peri-intubation patients with high shock indices may decrease the risk of peri-intubation cardiac arrest (Table). In patients deemed fluid intolerant or vasoplegic, early vasopressors should be administered to maintain perfusion pressure and vascular tone.

Traditionally, vasopressor infusions required immediate central venous access; however, recent studies show that peripherally infused vasopressors are low risk and reasonable alternatives to central infusions when given for short durations.[66,67] Norepinephrine is the preferred vasopressor in critically ill patients and should be started as a continuous infusion in patients with preintubation hypotension or shock.[68] Phenylephrine is a pure vasoconstrictor and increases vascular resistance and thereby blood pressure, but without associated inotropy, the increased blood pressure is at the cost of a lower cardiac output.[69] Dilute phenylephrine boluses (eg, 100 µg/mL) may be helpful to address hypotension induced by anesthetic agents to maintain systemic vascular resistance and diastolic perfusion of the coronary arteries until transient hypotension resolves or fluid resuscitation can be optimized; however, patients with a high SI or hypotension should be started on a continuous infusion of vasopressors with inotropic properties (eg, norepinephrine; Table).[70] Bolus doses of epinephrine can be effective, as well, when short-term vasoconstriction and inotropy are desired.

Laryngoscopy and tracheal intubation cause a sympathetic response, which may induce myocardial or cerebral malperfusion in ICU patients with little physiologic reserve. Most induction agents are sedative hypnotics without analgesia.[71] Propofol and benzodiazepines have sympatholytic effects that lead to myocardial depression and decreased vascular tone.[72]

Etomidate is hemodynamically neutral with little effect on myocardial contractility.[73,74] There are concerns about development of adrenal insufficiency in patients with sepsis; however, there is little evidence of harm when etomidate is used solely for induction.[75,76] Induction agent dose reductions to as low as 10% of standard dosing is reasonable in patients with shock or a high SI,[77,78] but should not replace adequate preintubation resuscitation.

Ketamine is an attractive induction agent in hypotension because of its sympathomimetic properties,[79] and has an overall complication rate similar to etomidate.[80] Jabre et al[79] compared etomidate and ketamine in 2009 and found no difference in serious complications for emergency intubation in septic patients. However, ketamine is also associated with laryngospasm, increased airway secretions, myocardial depression, increased cardiac output leading to myocardial ischemia and even cardiac arrest.[54]

Recommendations

1. Patients should have intravenous access sufficient for rapid fluid administration before intubation. 100% agreement (SD 0%)
2. Patients should be screened for high risk of hemodynamic collapse with intubation. Those with a SI >0.7 are at increased risk. 99% agreement (SD 1.5%)
3. Hypotensive patients due to obstructive shock secondary to acute or acute-on-chronic RV failure should be managed per the RV failure guidelines. 99% agreement (SD 3%)
4. Fluid-responsive and fluid-tolerant patients should be fluid resuscitated before intubation, or at least during the intubation attempt. 99.5% agreement (SD 1.5%)
5. When possible, vasopressor infusions should be started before intubation in patients that are not volume responsive or fluid tolerant. 99.5% agreement (SD 1.5%)
6. When vasopressor infusions are not possible, bolus-dosed vasopressors should be available and used to maintain systemic pressure during and after the intubation, until an infusion can be started. 100% agreement (SD 0%)
7. When bolus-dosed vasopressors are used, diluted epinephrine should be considered as the vasopressor of choice in patients with depressed myocardial function. 97% agreement (SD 5.6%)
8. Hemodynamically neutral induction agents should be used. 100% agreement (SD 0%)

Special Circumstances

RV Dysfunction. Emergency airway management in the critically ill patient requires consideration of the heart-lung interactions that characterize RV physiology interacting with venous return, pulmonary vascular resistance, and the cardiac output of the left ventricle (LV). In patients with chronic pulmonary arterial hypertension, pulmonary embolism, or acute RV failure, the exacerbation of RV performance with intubation can be the precipitating event for cardiac arrest. Thus, one must be able to diagnose and manage RV failure during airway management to mitigate risk of precipitating unfavorable hemodynamics or cardiac arrest with intubation.

RV dysfunction is characterized by the reduced ability of the right heart to provide adequate blood flow through the pulmonary circulation at a normal central venous pressure.[81] It is caused by any pathophysiologic process that reduces myocardial contractility or increases pulmonary vascular resistance and RV afterload.[82] The reduced downstream cardiac output is characterized by decreased LV end-diastolic volume. In a simplified manner, these downstream hemodynamic effects of RV pressure overload can be acutely worsened with the addition of positive intrathoracic pressure from mechanical ventilation, or volume overload from aggressive fluid resuscitation.

RV dysfunction often is characterized by RV dilation and reduced RV systolic longitudinal displacement. The RV is particularly sensitive to changes in afterload.[83] RV stroke volume decreases sharply (roughly 30%) with a 20 mm Hg pressure afterload increase; in contrast, a 20 mm Hg increase in LV afterload only drops LV stroke volume by roughly 10%.[82,83] Increased afterload eventually reaches a critical point in RV wall tension that leads to RV dilation, incompetence of the tricuspid valve with tricuspid regurgitation, and precipitates a downward spiral of ischemia—potentiating right and left ventricular failure via ventricular interdependence.[81]

Once RV dysfunction has been identified, any potential for improvement in RV function with increased preload should be closely scrutinized (Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206). Traditional dynamic methods of evaluating volume responsiveness by evaluating LV stroke volume swings with changes in intrathoracic pressure can be misleading because the RV is always preload responsive in the setting of RV dysfunction. Thus, volume responsiveness of the RV, not the LV, needs to be evaluated. When the RV is preload responsive, fluid boluses should be small (250 mL) to avoid rapid volume overload. If the RV is volume overloaded, aggressive diuresis may be beneficial before induction for improvement in RV hemodynamics. Norepinephrine may improve ventricular systolic function without increasing pulmonary vascular resistance.[83]

For clinical situations when the RV is not preload responsive or volume overloaded, afterload reduction

becomes the only remaining therapeutic option. This can be accomplished by treating any hypoxemia, atelectasis, and hypercapnia that all independently increase pulmonary vascular resistance. Inhaled pulmonary vasodilators are available to reduce the RV afterload and can be given without significant logistical difficulty (Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206). The major side effects of these include systemic hypotension and worsening of V/Q mismatch.[84]

Recommendations

1. Patients should be screened for significant RV dysfunction before intubation given the risk of decompensation with the transition to positive pressure ventilation. 94% agreement (SD 11%)
2. When RV dysfunction is present, patients should be evaluated for RV systolic function, and fluid and vasopressor tolerance. Empiric fluid resuscitation without this evaluation can further reduce RV function. 98% agreement (SD 4%)
3. Fluid and vasopressor tolerant patients should be resuscitated accordingly. 99.9% agreement (SD 0.3%)
4. Fluid-intolerant patients should have RV afterload reduced with inhaled or intravenous pulmonary vasodilators. 96% agreement (SD 8%)
5. Preintubation extracorporeal membrane oxygenation (ECMO) cannulation should be considered if available in patients with RV failure–induced shock. 98% agreement (SD 6%)
6. Preintubation diuresis should be considered in patients with RV volume overload. 100% agreement (SD 0%)
7. Hypercapnia should be avoided. 97% agreement (SD 8%)
8. Mean arterial pressure should be augmented to maintain coronary perfusion pressure. In patients with chronic pulmonary arterial hypertension, a higher mean arterial pressure should be targeted to keep mean arterial pressure > mean pulmonary artery pressure. 100% agreement (SD 0%)
9. Ventilation strategy after intubation should include a low mean airway pressure and a higher PEEP to avoid atelectasis. 94% agreement (SD 15.7%)

Severe Metabolic Acidosis

Carbon dioxide dissolved in the blood is cleared through alveolar ventilation. Therefore, reduction in ventilatory drive, neuromuscular inefficiency (increased ventilator load or reduced effort), or increased dead space will lead to an increase in CO_2 and a reduced pH. This respiratory acidosis can be corrected with an improvement in alveolar ventilation, generally in a 1:1 relationship where doubling the alveolar ventilation will reduce the CO_2 by half. BVM, NIPPV, or mechanical ventilation rapidly reduces inspiratory work of breathing and overcomes neuromuscular weakness to correct respiratory acidosis.[10]

In ventilatory failure due to metabolic acidosis, however, the bicarbonate buffering system is overwhelmed and compensatory removal of CO_2 through increased alveolar ventilation reaches a plateau. Severe metabolic acidosis such as diabetic ketoacidosis, salicylate or metformin toxicity, and severe lactic acidosis outpace the respiratory compensation attempts to keep blood pH in a normal range. Any reduced ventilatory compensation leads to a rapidly downward spiral of worsening acidosis and cardiopulmonary arrest.[85] Thus, intubating these patients with severe acidemia from an uncompensated metabolic acidosis can be particularly troublesome. It is challenging to match the ventilatory requirement of the patient with the capacity of the mechanical ventilator especially in the context of the apneic period during induction.

Before intubation, preoxygenation with NIPPV will allow for a reduction in work of breathing and will provide an estimate of the minute ventilation requirement to maintain the profoundly acidemic state. If the minute ventilation requirement is higher than can be safely achieved in a passively breathing patient, then one should consider avoiding rapid sequence intubation and especially long-acting paralytics. An awake approach with a spontaneously breathing patient and the use of a spontaneous breathing (ie, pressure supported) mode of mechanical ventilation may be the option of choice for safely managing these airways. The use of a high-flow NC system that washes out dead space and improves ventilation may be incredibly beneficial to prevent a respiratory acidosis component during the procedure (Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206). Preintubation bicarbonate boluses in patients with very high minute ventilation are controversial, lack data, and had a low rate of agreement among the workgroup 77% agreement (SD 37%).

Recommendations

1. Patients with severe metabolic acidosis are at high risk of decompensation due to volume depletion and inadequate alveolar ventilation after intubation. 100% agreement (SD 0%)
2. Patients with high minute ventilation requirements should be considered for awake intubation to maintain spontaneous respiration. 99% agreement (SD 3%)
3. Consider a spontaneous breathing mode after intubation in patients with very high minute ventilation requirements. 99% agreement (SD 3%)

Neurologically Injured Patients

It is imperative to maintain eucapnia and normoxia during intubation in the neurologically compromised patient, because cerebral blood flow is very sensitive to changes in CO_2 and oxygen saturations.[86] During induction, extremes of mean arterial pressure, intracranial pressure (ICP), and subsequent variability in cerebral perfusion pressure should be prevented. Maintenance of stable hemodynamics in the severe traumatic brain injury patient is critical for adequate cerebral perfusion (cerebral perfusion pressure = mean arterial pressure [MAP] – ICP). The brain is able to autoregulate perfusion over a range of cerebral perfusion pressure from 50 to 100 mm Hg. However, severe traumatic brain injury affects the ability to autoregulate.

Severe traumatic brain injury is a common process complicating airway patency in the prehospital environment often necessitating endotracheal intubation. Unfortunately, laryngoscopy and endotracheal intubation are noxious stimuli precipitating sympathetic discharge and a significant cardiovascular response. Pretreatment with lidocaine and opiates, such as fentanyl, have limited evidence to strongly support out-of-OR use in acute severe traumatic brain injury.[87] Much of the data is either in small OR patient cohorts or is in elective surgery or brain tumor populations.[88,89] Topical application of 4% lidocaine may be of use; however, the data are limited without randomized, blinded trials.

Esmolol has also been investigated as a potential quick-acting pharmacologic intervention in the management of the out-of-OR airway. Prospective study by Ugur et al[90] in 2007 found that esmolol pretreatment could control tachycardia and rate-pressure product markers of hemodynamics when compared to lidocaine. Chung et al[91] in 1992 noted a potential synergistic effect of esmolol and fentanyl to attenuate hemodynamic response greater than either agent alone (fentanyl 2 μg/kg or esmolol 2 mg/kg).[91]

In addition to pharmacologic pretreatment approaches, the choice and dose of a hemodynamically neutral induction agent are particularly important in the physiologic management of the neurocritical care patient. While ketamine was previously thought to have negative impacts on ICP, this dogma has since been overturned (Supplemental Digital Content, Table 1, http://links.lww.com/AA/D206).

Recommendations

1. Eucapnia should be maintained before, during, and after intubation. 99.9% agreement (SD 0.3%)
2. Hemodynamically neutral induction agents should be used. 100% agreement (SD 0%)
3. Patients should be positioned 30° upright, when possible. 100% agreement (SD 0%)
4. Postintubation management should include limiting PEEP to promote cerebral venous drainage. 92.5% agreement (SD 23.7%)

DISCUSSION

As more data rapidly emerge on the physiology of endotracheal intubation, there exists a need for focused guidelines on the heart-lung interactions and the pharmacologic interventions that underpin airway management. The above guidelines serve to show the general level of agreement for various physiologic tenets of airway management among a diverse group of specialists specific to the out-of-OR environment. Although randomized trials may not necessarily show statistically significant outcomes with particular well-regarded interventions (eg, Semler et al[35] with apneic oxygenation), these SAM guidelines reflect the sentiment and practice patterns of practicing airway specialists. The diverse backgrounds within the working group provide a variety of perspectives on the utility of various interventions within the resource limitations of the out-of-OR environment.

Limitations of the guidelines are the lack of a risk of bias assessment. Furthermore, no librarian formalized the literature search process. However, because most of the recommendations have limited to no data in published literature, we decided against a traditional Delphi process and instead sent out the list to the SAM Committee to generate their level of agreement with each recommendation.

The multidisciplinary approach of SAM provides an avenue for discourse that transcends subspecialty and cultural barriers. Because the various recommendations from the Committee are diverse and expansive in breadth, the top 10 recommendations from the multidisciplinary group are summarized in the Table. By highlighting themes in the context of a physiologic approach, these SAM guidelines for the physiologically difficult airway define principles that extend beyond a single practice environment and highlight areas where research is needed. ■

DISCLOSURES

Name: Rebecca L. Kornas, MD.
Contribution: This author helped with the study design/concept, acquisition of data, analysis of data, drafting the manuscript, and critical manuscript revision.
Conflicts of Interest: None.
Name: Clark G. Owyang, MD.
Contribution: This author helped with the study design/concept, acquisition of data, analysis of data, drafting the manuscript, and critical manuscript revision.
Conflicts of Interest: None.

Name: John C. Sakles, MD.
Contribution: This author helped with the study design/concept, analysis of data, and critical manuscript revision.
Conflicts of Interest: J. C. Sakles has previously served as a consultant for Verathon Inc (Bothwell, WA).
Name: Lorraine J. Foley, MD.
Contribution: This author helped with the study design/concept, analysis of data, and critical manuscript revision.
Conflicts of Interest: L. J. Foley is a paid consultant for Vyaire Medical.
Name: Jarrod M. Mosier, MD.
Contribution: This author helped with the study design/concept, acquisition of data, analysis of data, drafting the manuscript, and critical manuscript revision.
Conflicts of Interest: J. M. Mosier previously served as a consultant Vyaire, and is a consultant for Verathon, Inc.
This manuscript was handled by: Narasimhan Jagannathan, MD, MBA.

CONTRIBUTORS

Thomas Terndrup, MD; Art French, MD; Richard Galgon, MD; Thomas Friedland, MD; Jessica Feinleib, MD, PhD; David J. Olvera; Chivas Guillote, RN; Drew Fuller, MD; Carin Hagberg, MD; Monique Steffani; Lynette Mark, MD; Clark G. Owyang, MD.

REFERENCES

1. Edelman DA, Perkins EJ, Brewster DJ. Difficult airway management algorithms: a directed review. *Anaesthesia.* 2019;74:1175–1185.
2. Frerk C, Mitchell VS, McNarry AF, et al; Difficult Airway Society intubation guidelines working group. Difficult Airway Society 2015 guidelines for management of unanticipated difficult intubation in adults. *Br J Anaesth.* 2015;115:827–848.
3. Higgs A, McGrath BA, Goddard C, et al; Difficult Airway Society; Intensive Care Society; Faculty of Intensive Care Medicine; Royal College of Anaesthetists. Guidelines for the management of tracheal intubation in critically ill adults. *Br J Anaesth.* 2018;120:323–352.
4. Apfelbaum JL, Hagberg CA, Caplan RA, et al; American Society of Anesthesiologists Task Force on Management of the Difficult Airway. Practice guidelines for management of the difficult airway: an updated report by the American Society of Anesthesiologists Task Force on Management of the Difficult Airway. *Anesthesiology.* 2013;118:251–270.
5. Rui PKK. *National Hospital Ambulatory Medical Care Survey: 2014 Emergency Department Summary Tables*. Atlanta, GA: Centers for Disease Control and Prevention; 2014.
6. Pfuntner A, Wier LM, Stocks C. Most Frequent Procedures Performed in U.S. Hospitals, 2011: Statistical Brief #165. Healthcare Cost and Utilization Project (HCUP) Statistical Briefs. Rockville, MD: Agency for Healthcare Research and Quality; 2013.
7. Natt BS, Malo J, Hypes CD, Sakles JC, Mosier JM. Strategies to improve first attempt success at intubation in critically ill patients. *Br J Anaesth.* 2016;117 (suppl 1):i60–i68.
8. De Jong A, Rolle A, Molinari N, et al. Cardiac arrest and mortality related to intubation procedure in critically ill adult patients: a Multicenter Cohort Study. *Crit Care Med.* 2018;46:532–539.
9. Mosier JM. Physiologically difficult airway in critically ill patients: winning the race between haemoglobin desaturation and tracheal intubation. *Br J Anaesth.* 2020;125:e1–e4.
10. Mosier JM, Joshi R, Hypes C, Pacheco G, Valenzuela T, Sakles JC. The physiologically difficult airway. *West J Emerg Med.* 2015;16:1109–1117.
11. Davis DP, Dunford JV, Poste JC, et al. The impact of hypoxia and hyperventilation on outcome after paramedic rapid sequence intubation of severely head-injured patients. *J Trauma.* 2004;57:1–8.
12. Mort TC. The incidence and risk factors for cardiac arrest during emergency tracheal intubation: a justification for incorporating the ASA Guidelines in the remote location. *J Clin Anesth.* 2004;16:508–516.
13. Davis DP, Lemieux J, Serra J, Koenig W, Aguilar SA. Preoxygenation reduces desaturation events and improves intubation success. *Air Med J.* 2015;34:82–85.
14. Mort TC. Emergency tracheal intubation: complications associated with repeated laryngoscopic attempts. *Anesth Analg.* 2004;99:607–613.
15. Griesdale DE, Bosma TL, Kurth T, Isac G, Chittock DR. Complications of endotracheal intubation in the critically ill. *Intensive Care Med.* 2008;34:1835–1842.
16. Jaber S, Jung B, Corne P, et al. An intervention to decrease complications related to endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Intensive Care Med.* 2010;36:248–255.
17. Sakles JC. Maintenance of oxygenation during rapid sequence intubation in the emergency department. *Acad Emerg Med.* 2017;24:1395–1404.
18. Baraka AS, Taha SK, Aouad MT, El-Khatib MF, Kawkabani NI. Preoxygenation: comparison of maximal breathing and tidal volume breathing techniques. *Anesthesiology.* 1999;91:612–616.
19. Berry CB, Myles PS. Preoxygenation in healthy volunteers: a graph of oxygen "washin" using end-tidal oxygraphy. *Br J Anaesth.* 1994;72:116–118.
20. Campbell IT, Beatty PC. Monitoring preoxygenation. *Br J Anaesth.* 1994;72:3–4.
21. Groombridge C, Chin CW, Hanrahan B, Holdgate A. Assessment of common preoxygenation strategies outside of the operating room environment. *Acad Emerg Med.* 2016;23:342–346.
22. Driver BE, Prekker ME, Kornas RL, Cales EK, Reardon RF. Flush rate oxygen for emergency airway preoxygenation. *Ann Emerg Med.* 2017;69:1–6.
23. Driver BE, Klein LR, Carlson K, Harrington J, Reardon RF, Prekker ME. Preoxygenation with flush rate oxygen: comparing the nonrebreather mask with the bag-valve mask. *Ann Emerg Med.* 2018;71:381–386.
24. Hayes-Bradley C, Lewis A, Burns B, Miller M. Efficacy of nasal cannula oxygen as a preoxygenation adjunct in emergency airway management. *Ann Emerg Med.* 2016;68:174–180.
25. Pavlov I, Medrano S, Weingart S. Apneic oxygenation reduces the incidence of hypoxemia during emergency intubation: a systematic review and meta-analysis. *Am J Emerg Med.* 2017;35:1184–1189.
26. Papazian L, Corley A, Hess D, et al. Use of high-flow nasal cannula oxygenation in ICU adults: a narrative review. *Intensive Care Med.* 2016;42:1336–1349.
27. Frumin MJ, Epstein RM, Cohen G. Apneic oxygenation in man. *Anesthesiology.* 1959;20:789–798.
28. Mosier JM, Hypes CD, Sakles JC. Understanding preoxygenation and apneic oxygenation during intubation in the critically ill. *Intensive Care Med.* 2017;43:226–228.
29. Ramachandran SK, Cosnowski A, Shanks A, Turner CR. Apneic oxygenation during prolonged laryngoscopy in obese patients: a randomized, controlled trial of nasal oxygen administration. *J Clin Anesth.* 2010;22:164–168.
30. Wimalasena Y, Burns B, Reid C, Ware S, Habig K. Apneic oxygenation was associated with decreased desaturation rates during rapid sequence intubation by an Australian

helicopter emergency medicine service. *Ann Emerg Med.* 2015;65:371–376.
31. Sakles JC, Mosier JM, Patanwala AE, Dicken JM. Apneic oxygenation is associated with a reduction in the incidence of hypoxemia during the RSI of patients with intracranial hemorrhage in the emergency department. *Intern Emerg Med.* 2016;11:983–992.
32. Brainard A, Chuang D, Zeng I, Larkin GL. A randomized trial on subject tolerance and the adverse effects associated with higher- versus lower-flow oxygen through a standard nasal cannula. *Ann Emerg Med.* 2015;65:356–361.
33. Patel A, Nouraei SA. Transnasal Humidified Rapid-Insufflation Ventilatory Exchange (THRIVE): a physiological method of increasing apnoea time in patients with difficult airways. *Anaesthesia.* 2015;70:323–329.
34. Fraioli RL, Sheffer LA, Steffenson JL. Pulmonary and cardiovascular effects of apneic oxygenation in man. *Anesthesiology.* 1973;39:588–596.
35. Semler MW, Janz DR, Lentz RJ, et al; FELLOW Investigators; Pragmatic Critical Care Research Group. Randomized trial of apneic oxygenation during endotracheal intubation of the critically ill. *Am J Respir Crit Care Med.* 2016;193:273–280.
36. Caputo N, Azan B, Domingues R, et al; Lincoln Airway Group. Emergency department use of apneic oxygenation versus usual care during rapid sequence intubation: a randomized controlled trial (The ENDAO Trial). *Acad Emerg Med.* 2017;24:1387–1394.
37. Mort TC. Preoxygenation in critically ill patients requiring emergency tracheal intubation. *Crit Care Med.* 2005;33:2672–2675.
38. Braude D, Richards M. Rapid Sequence Airway (RSA)–a novel approach to prehospital airway management. *Prehosp Emerg Care.* 2007;11:250–252.
39. Das B, Mitra S, Jamil SN, Varshney RK. Comparison of three supraglottic devices in anesthetised paralyzed children undergoing elective surgery. *Saudi J Anaesth.* 2012;6:224–228.
40. Spoletini G, Alotaibi M, Blasi F, Hill NS. Heated humidified high-flow nasal oxygen in adults: mechanisms of action and clinical implications. *Chest.* 2015;148:253–261.
41. Parke RL, Eccleston ML, McGuinness SP. The effects of flow on airway pressure during nasal high-flow oxygen therapy. *Respir Care.* 2011;56:1151–1155.
42. Baillard C, Fosse JP, Sebbane M, et al. Noninvasive ventilation improves preoxygenation before intubation of hypoxic patients. *Am J Respir Crit Care Med.* 2006;174:171–177.
43. El-Khatib MF, Kanazi G, Baraka AS. Noninvasive bilevel positive airway pressure for preoxygenation of the critically ill morbidly obese patient. *Can J Anaesth.* 2007;54:744–747.
44. Mani NS, Budak JZ, Lan KF, et al. Prevalence of COVID-19 infection and outcomes among symptomatic healthcare workers in Seattle, Washington. *Clin Infect Dis*. Published online ahead of print June 16, 2020. doi: 10.1093/cid/ciaa761.
45. Schwartz J, King CC, Yen MY. Protecting healthcare workers during the coronavirus disease 2019 (COVID-19) outbreak: lessons from Taiwan's severe acute respiratory syndrome response. *Clin Infect Dis.* 2020;71:858–860.
46. Ferioli M, Cisternino C, Leo V, Pisani L, Palange P, Nava S. Protecting healthcare workers from SARS-CoV-2 infection: practical indications. *Eur Respir Rev*. 2020;29:200068.
47. Orser BA. Recommendations for endotracheal intubation of COVID-19 patients. *Anesth Analg.* 2020;130:1109–1110.
48. Miller DC, Beamer P, Billheimer D, et al. Aerosol risk with noninvasive respiratory support in patients with COVID-19. *J Am Coll Emerg Physicians Open*. 2020;1:521–526.
49. Brown CA III, Mosier JM, Carlson JN, Gibbs MA. Pragmatic recommendations for intubating critically ill patients with suspected COVID-19. *J Am Coll Emerg Physicians Open*. 2020;1:80–84.
50. Dixon BJ, Dixon JB, Carden JR, et al. Preoxygenation is more effective in the 25 degrees head-up position than in the supine position in severely obese patients: a randomized controlled study. *Anesthesiology.* 2005;102:1110–1115.
51. Semler MW, Janz DR, Russell DW, et al; Check-UP Investigators(∗); Pragmatic Critical Care Research Group. A multicenter, randomized trial of ramped position vs sniffing position during endotracheal intubation of critically ill adults. *Chest.* 2017;152:712–722.
52. Weingart SD, Trueger NS, Wong N, Scofi J, Singh N, Rudolph SS. Delayed sequence intubation: a prospective observational study. *Ann Emerg Med.* 2015;65:349–355.
53. Driver BE, Reardon RF. Apnea after low-dose ketamine sedation during attempted delayed sequence intubation. *Ann Emerg Med.* 2017;69:34–35.
54. Strayer RJ, Nelson LS. Adverse events associated with ketamine for procedural sedation in adults. *Am J Emerg Med.* 2008;26:985–1028.
55. Jaber S, Amraoui J, Lefrant JY, et al. Clinical practice and risk factors for immediate complications of endotracheal intubation in the intensive care unit: a prospective, multiple-center study. *Crit Care Med.* 2006;34:2355–2361.
56. Schwartz DE, Matthay MA, Cohen NH. Death and other complications of emergency airway management in critically ill adults. A prospective investigation of 297 tracheal intubations. *Anesthesiology.* 1995;82:367–376.
57. Manley G, Knudson MM, Morabito D, Damron S, Erickson V, Pitts L. Hypotension, hypoxia, and head injury: frequency, duration, and consequences. *Arch Surg.* 2001;136:1118–1123.
58. Green R, Hutton B, Lorette J, Bleskie D, McIntyre L, Fergusson D. Incidence of postintubation hemodynamic instability associated with emergent intubations performed outside the operating room: a systematic review. *CJEM.* 2014;16:69–79.
59. Heffner AC, Swords D, Kline JA, Jones AE. The frequency and significance of postintubation hypotension during emergency airway management. *J Crit Care.* 2012;27:417 e419–413 e419.
60. Green RS, Turgeon AF, McIntyre LA, et al; Canadian Critical Care Trials Group (CCCTG). Postintubation hypotension in intensive care unit patients: a multicenter cohort study. *J Crit Care.* 2015;30:1055–1060.
61. Rady MY, Rivers EP, Nowak RM. Resuscitation of the critically ill in the ED: responses of blood pressure, heart rate, shock index, central venous oxygen saturation, and lactate. *Am J Emerg Med.* 1996;14:218–225.
62. Walls RM, Brown CA III, Bair AE, Pallin DJ; NEAR II Investigators. Emergency airway management: a multicenter report of 8937 emergency department intubations. *J Emerg Med.* 2011;41:347–354.
63. Heffner AC, Swords DS, Neale MN, Jones AE. Incidence and factors associated with cardiac arrest complicating emergency airway management. *Resuscitation.* 2013;84:1500–1504.
64. Kim WY, Kwak MK, Ko BS, et al. Factors associated with the occurrence of cardiac arrest after emergency tracheal intubation in the emergency department. *PLoS One.* 2014;9:e112779.
65. Wardi G, Villar J, Nguyen T, et al. Factors and outcomes associated with inpatient cardiac arrest following emergent endotracheal intubation. *Resuscitation.* 2017;121:76–80.
66. Loubani OM, Green RS. A systematic review of extravasation and local tissue injury from administration of vasopressors through peripheral intravenous catheters and central venous catheters. *J Crit Care.* 2015;30:653.e9–653.17.
67. Ricard JD, Salomon L, Boyer A, et al. Central or peripheral catheters for initial venous access of ICU patients: a randomized controlled trial. *Crit Care Med.* 2013;41:2108–2115.

68. Monnet X, Letierce A, Hamzaoui O, et al. Arterial pressure allows monitoring the changes in cardiac output induced by volume expansion but not by norepinephrine. *Crit Care Med.* 2011;39:1394–1399.
69. Funk DJ, Jacobsohn E, Kumar A. The role of venous return in critical illness and shock-part I: physiology. *Crit Care Med.* 2013;41:255–262.
70. Panchal AR, Satyanarayan A, Bahadir JD, Hays D, Mosier J. Efficacy of bolus-dose phenylephrine for peri-intubation hypotension. *J Emerg Med.* 2015;49:488–494.
71. Hagberg CA. *Benumof and Hagberg's Airway Management*. 3rd ed. Saunders; 2012.
72. Ebert TJ, Muzi M, Berens R, Goff D, Kampine JP. Sympathetic responses to induction of anesthesia in humans with propofol or etomidate. *Anesthesiology.* 1992;76:725–733.
73. Tassani P, Martin K, Jänicke U, Ott E. Bolus administration of eltanolone, thiopental, or etomidate does not affect systemic vascular resistance during cardiopulmonary bypass. *J Cardiothorac Vasc Anesth.* 1997;11:562–564.
74. Morris C, Perris A, Klein J, Mahoney P. Anaesthesia in haemodynamically compromised emergency patients: does ketamine represent the best choice of induction agent? *Anaesthesia.* 2009;64:532–539.
75. Gu WJ, Wang F, Tang L, Liu JC. Single-dose etomidate does not increase mortality in patients with sepsis: a systematic review and meta-analysis of randomized controlled trials and observational studies. *Chest.* 2015;147:335–346.
76. Chan CM, Mitchell AL, Shorr AF. Etomidate is associated with mortality and adrenal insufficiency in sepsis: a meta-analysis*. *Crit Care Med.* 2012;40:2945–2953.
77. Reich DL, Hossain S, Krol M, et al. Predictors of hypotension after induction of general anesthesia. *Anesth Analg.* 2005;101:622–628.
78. Heffner AC, Swords DS, Nussbaum ML, Kline JA, Jones AE. Predictors of the complication of postintubation hypotension during emergency airway management. *J Crit Care.* 2012;27:587–593.
79. Jabre P, Combes X, Lapostolle F, et al; KETASED Collaborative Study Group. Etomidate versus ketamine for rapid sequence intubation in acutely ill patients: a multicentre randomised controlled trial. *Lancet.* 2009;374:293–300.
80. Patanwala AE, McKinney CB, Erstad BL, Sakles JC. Retrospective analysis of etomidate versus ketamine for first-pass intubation success in an academic emergency department. *Acad Emerg Med.* 2014;21:87–91.
81. Greyson CR. Pathophysiology of right ventricular failure. *Crit Care Med.* 2008;36:S57–S65.
82. Grignola JC, Domingo E. Acute right ventricular dysfunction in intensive care unit. *Biomed Res Int.* 2017;2017:8217105.
83. Ventetuolo CE, Klinger JR. Management of acute right ventricular failure in the intensive care unit. *Ann Am Thorac Soc.* 2014;11:811–822.
84. Harjola VP, Mebazaa A, Čelutkienė J, et al. Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology. *Eur J Heart Fail.* 2016;18:226–241.
85. Manthous CA. Avoiding circulatory complications during endotracheal intubation and initiation of positive pressure ventilation. *J Emerg Med.* 2010;38:622–631.
86. Bucher J, Koyfman A. Intubation of the neurologically injured patient. *J Emerg Med.* 2015;49:920–927.
87. Hassani V, Movassaghi G, Goodarzi V, Safari S. Comparison of fentanyl and fentanyl plus lidocaine on attenuation of hemodynamic responses to tracheal intubation in controlled hypertensive patients undergoing general anesthesia. *Anesth Pain Med.* 2013;2:115–118.
88. Perkins ZB, Wittenberg MD, Nevin D, Lockey DJ, O'Brien B. The relationship between head injury severity and hemodynamic response to tracheal intubation. *J Trauma Acute Care Surg.* 2013;74:1074–1080.
89. Samaha T, Ravussin P, Claquin C, Ecoffey C. [Prevention of increase of blood pressure and intracranial pressure during endotracheal intubation in neurosurgery: esmolol versus lidocaine]. *Ann Fr Anesth Reanim.* 1996;15:36–40.
90. Ugur B, Ogurlu M, Gezer E, Nuri Aydin O, Gürsoy F. Effects of esmolol, lidocaine and fentanyl on haemodynamic responses to endotracheal intubation: a comparative study. *Clin Drug Investig.* 2007;27:269–277.
91. Chung KS, Sinatra RS, Halevy JD, Paige D, Silverman DG. A comparison of fentanyl, esmolol, and their combination for blunting the haemodynamic responses during rapid-sequence induction. *Can J Anaesth.* 1992;39:774–779.